



A guide to cranial nerve testing for musculoskeletal clinicians

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ABSTRACT

Background: Neurological examination in musculoskeletal practice is a key element of safe and appropriate orthopedic clinical practice. With physiotherapists currently positioning themselves as advanced first line practitioners, it is essential that those who treat patients who present with neck/head/orofacial pain and associated symptoms, should have an index of suspicion of cranial nerve (CN) dysfunction. They should be able to examine and determine if CN dysfunction is present, and make appropriate clinical decisions based upon those findings. **Methods:** This paper summarizes the functions, potential impairments of the nerves, associated conditions, and basic skills involved in cranial nerve examination. **Results:** A summary of cranial nerve examination is provided, which is based on the function of the nerves. This is intended to facilitate clinicians to feel more confident at understanding neural function/impairment, as well as performing and interpreting the examination. **Conclusion:** This paper illustrates that CN testing can be performed quickly, efficiently and without the need for complicated or potentially unavailable equipment. An understanding of the CN's function and potential reasons for impairment is likely to increase the frequency of CN testing in orthopedic clinical practice and referral if positive findings are encountered.

KEYWORDS

Cranial nerves; impairment; examination; musculoskeletal practice

With physiotherapy clinicians worldwide, assuming first contact roles as 'advanced clinical practitioners' (ACP), there is a need for relevant training, knowledge, clinical reasoning, differential diagnosis and triage skills. Above all, it is essential that clinicians have the ability to recognize the key elements of the patient history, and the physical examination which may be indicative of serious pathology or a potential risk of serious adverse events [1]. As such, the ability to perform a complete neurological work up is paramount.

Neurological examination in musculoskeletal (MSK) practice has always been considered to be a key element of safe and appropriate clinical practice [2] and is commonly used to identify upper/lower limb and upper motor neuron involvement. However, for reasons as yet to be elucidated, it appears that undergraduate training in a range of countries has not adequately prepared a generation of physiotherapists in the rationale for and fundamentals of cranial nerve (CN) examination. Personal experience and recent Twitter polls of over 600 physiotherapists [3], have highlighted that many were either not taught, or do not feel confident in their knowledge, skills and clinical reasoning, with regard to the clinical application of CN examination. Although the scale and distribution of this knowledge deficit remains to be quantified in full, the authors are aware of as yet unpublished data from surveys in Italy and the UK, that are indicative of a shortfall in education and skills in this area of

neurological examination. However to date, there is no published data on the scale of the issue.

Neck pain and associated symptoms are common conditions that lead to pain, impairments, disability and a considerable economic burden [4]. The association between neck pain and headache manifestations including, facial pain, jaw pain, muscular dysfunction, visual/balance disturbances, and dizziness has been well documented and debated in the physiotherapy world for decades in a range of domains [5–7].

Patients commonly seek physiotherapy assessment and intervention for neck pain/stiffness and headache. It is well documented that neck pain, headache and orofacial pain are commonly reported as the early signs of arterial dissection leading to stroke [8,9]. Indeed it has been suggested that neck pain and headache may precede the onset of obvious frank neurological symptoms for as long as 14 days [10], thought to be a more obvious factor in clinical reasoning errors associated with major adverse events (MAE). Subtle CN palsy is a known pre-ischemic feature of carotid artery dissection due to anatomical proximity of the lower cranial nerves (IX, X and XII) to the carotid sheath. Lower CN lesions should be considered in cases of neck pain/head ache, neuralgic pain, disturbed speech, swallowing, coughing, deglutition, sensory dysfunctions, taste, or autonomic dysfunctions, dysphagia, pharyngeal pain, cardiac or gastrointestinal

Table 1. Cranial nerves and their functions (Diagonal lines – Sensory function – Smell/Hearing; Horizontal lines – Motor and sensory function of the eyes; Vertical lines – Motor and sensory function of the face/jaw/throat/tongue; Crossed lines – Motor function of the head/neck/shoulders).

Number	Name	Type	Function
I	Olfactory	Sensory	Smell (olfaction)
II	Optic	Sensory	Vision (acuity and field)
III	Oculomotor	Motor	Eye movements, elevation of eyelid. Pupil size and reactivity to light
IV	Trochlear	Motor	Eye movement (vertical and adduction)
V	Trigeminal	Mixed	Chewing, face/mouth sensation. Corneal reflex (sensory)
VI	Abducens	Motor	Eye movements – abduction
VII	Facial	Mixed	Facial expression, eyelid and lip closure, taste. Corneal reflex (motor)
VIII	Vestibulocochlear (auditory)	Sensory	Hearing, balance/equilibrium
IX	Glossopharyngeal	Mixed	Gagging, swallowing (sensory), taste
X	Vagus	Mixed	Gagging, swallowing (motor), speech (sound)
XI	Accessory	Motor	Head/neck/shoulder movement
XII	Hypoglossal	Motor	Tongue movement, speech (articulation)

compromise, or weakness of the trapezius, sternocleidomastoid, or the tongue muscles [11]. However, clinicians should note that there are multiple potential causes for CN impairment [12], and appropriate management requires an early recognition. It is the role of the astute clinician to be able to make sense of the complex presentations that commonly combine, associated with neck pain, with or without trauma. The key objective of any examination is to filter out those patients who may need referral for further examination or testing, either as urgent or non-urgent cases. It has been suggested that CN examination should be an integral part of that process [13].

In medicine, testing of the CN's has been documented and practised since the late 1800's [14] and is an integral part of a complete neurological examination. However, with the development of modern imaging, the clinical examination has been reduced [2]. Conversely, in physiotherapy, the suggestion is, that this is not routine and furthermore, there appears to be a perception that these skills are the domain of medical physicians, and somehow outside the skill set of physiotherapists. However, this notion does not fit with the intention for physiotherapists to achieve 'advanced practice level capabilities'.

Purpose

CN examination appears to be perceived as a challenging examination which may be time consuming, difficult to interpret and involves specialized equipment or skills e.g. fundoscopy. This article aims to provide clinicians with an introduction and updated guide to the CN's, placed in a clinical reasoning context, which allows the reader to understand the functions of

the cranial nerves, the rationale for, and application of appropriate CN examination in musculoskeletal cases.

The cranial nerves – what are they, what are their functions and why would you test them?

The cranial nerves are involved in sensations such as vision, smell, hearing, taste and facial sensation, as well as the functions of eye movement, head, face, neck/shoulder, jaw, tongue and throat functions such as speech, swallowing or gagging. As such, some of the CN's have motor functions, some have sensory functions, and some have mixed functions (see Table 1).

There are many reasons for neural impairment, including insidious mechanisms such as local pressure from space occupying lesions, inflammation, infection, atrophy, or demyelination. For clinicians, gaining a working knowledge of the individual nerve functions is key to understanding, performing and interpreting the testing [2]. Appendix 1 presents lists the functions of, and the more common reasons or mechanisms of impairment at each individual nerve.

A function based approach to CN testing

A basic CN testing can be performed easily and quickly in the clinic without the need for specialized equipment. A Snellen chart (or newspaper) together with a pen light or small flashlight, neurotips, cotton wool and tongue depressor (optional) are all that is required to perform the examination. It may be helpful to use a cheat sheet, in clinics where this type of testing is only performed occasionally.

Those who have learned the nerves in order may prefer to test that way. However, physiotherapists are well used to testing function, and for that reason it is logical, to consider grouping the tests together as shown in Table 2.

A step by step guide to testing the cranial nerves

In line with the current move toward Telehealth, the description of testing, has been written as an online Telehealth consultation wherever possible. Tests that additionally can be used in a face-to-face consultation are also included, so that clinicians can choose either option. The summary of the examination presented in Table 2 describes the use of the tests in Telehealth. Appendix 2 gives a summary of the test procedures in a face-to-face consultation.

Following the function based approach to testing outlined above, the examination may proceed as follows:

The senses of smell and hearing

CN I – Olfactory nerve

Test: Ask the patient to close their eyes and one nostril, introduce a familiar smell (soap/perfume/coffee) and ask the person to identify it (Figure 1). Repeat on the opposite side. Note any side to side differences.

CN VIII – Vestibulocochlear nerve

If you have completed a full subjective history prior to this point, the likelihood is that the patient can hear you.



Figure 1. CN I test (identify familiar smell).

Test: compare side to side hearing by asking the patient to rub their fingers together close to each ear. Note any side to side differences.

In a face-to-face consultation you can vigorously rub the fingers together near to each ear with the patient's eyes closed. If there is lateralization or hearing abnormalities, perform the Rinne and Weber tests using the 256-Hz tuning fork.

Rinne's test (Figure 2a and 2b): The tuning fork is struck and placed on the mastoid process. The patient is requested to indicate when the sound is no longer audible. As soon as the sound is extinguished, the tuning fork is placed next to the external auditory meatus to assess whether it can be heard. In a patient with normal hearing, air conduction should be greater than bone conduction, so the patient should be able to hear the tuning fork.

Weber's test (Figure 3): place the tuning fork in the middle of the forehead and the sound is heard from there. The sound should be heard equally on both ears.

The examination of the eyes – CN's II, III, IV and VI

Ask about any visual disturbances e.g. double vision.

Table 2. A function based approach to order and testing (Diagonal lines – Sensory function – Smell/Hearing; Horizontal lines – Motor and sensory function of the eyes; Vertical lines – Motor and sensory function of the face/jaw/throat/tongue; Crossed lines – Motor function of the head/neck/shoulders. OEMS = Extra ocular movements).

Number	Name	Examination
I	Olfactory	Identify a familiar smell (soap/perfume)
VIII	Vestibulocochlear (auditory)	Ask patient if they can hear fingers rubbing (close to ear) or whispered number sequence
II	Optic	Test each eye with Snellen chart or newspaper. Test visual fields in 4 quadrants
III	Oculomotor	Check pupil reaction to light (both should constrict). Check all OEMS (H-test). Check accommodation (finger to nose)
IV	Trochlear	H-Test – observe down and in
VI	Abducens	H-Test – observe side to side
V	Trigeminal	Test jaw strength (open mouth) – try to close/move laterally Check facial sensation – sharp/blunt Test corneal reflex
VII	Facial	Ask patient to smile, raise eyebrows, puff out cheeks. Check for symmetry. Ask about taste.
IX	Glossopharyngeal	Assess gag reflex with tongue depressor. Ask patient to swallow.
X	Vagus	Ask patient to say 'Aaaaaaaah', observe for symmetrical elevation of palate and uvula
XII	Hypoglossal	Patient protrudes tongue, check for deviation, look for fasciculations. Patient pushes out cheek with tongue, check power by pushing cheek.
XI	Accessory	Check resisted head rotation (sternocleidomastoid) and shoulder elevation (trapezius – upper fibers).

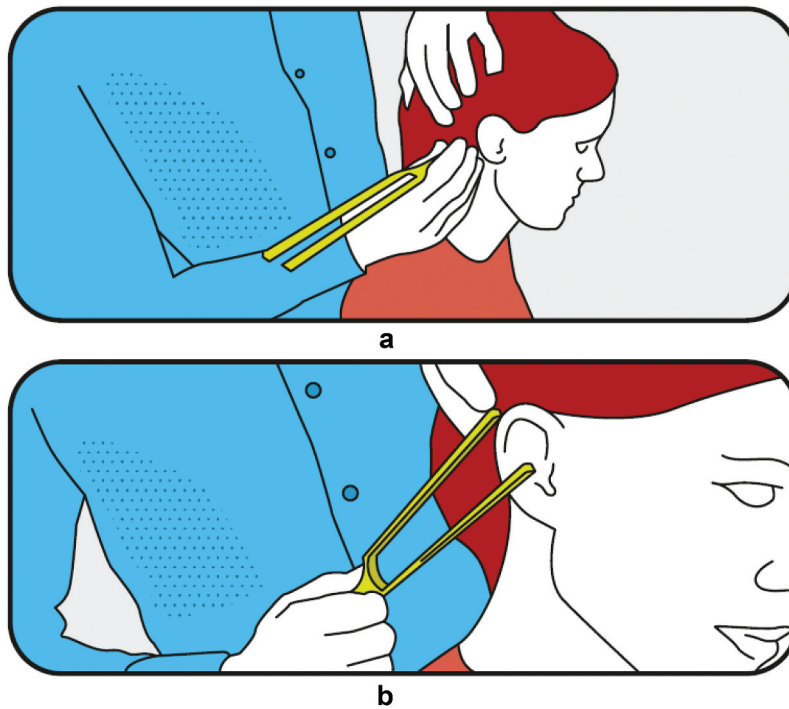


Figure 2. (a) Rinne's test (tuning fork is struck and placed on the mastoid process). (b) Rinne's test (fork is placed next to the external auditory meatus).

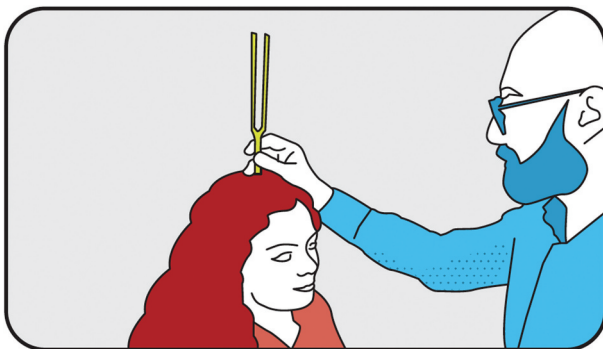


Figure 3. Weber's test (fork is placed on the vertex of the head).

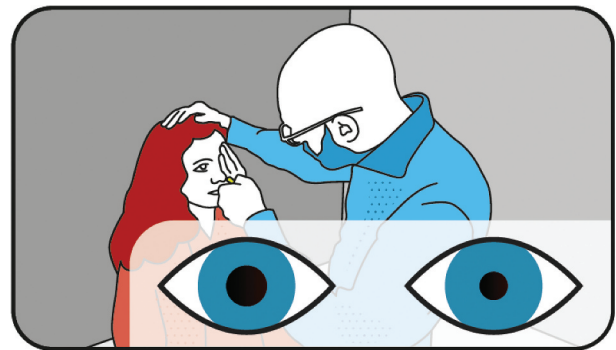


Figure 5. CN III test (pupil reaction to light).



Figure 4. CN II test (eye examination).

Observe the eyes: Look for size, shape and symmetry (eyelids, pupils).

CN II – Optic nerve.

Visual acuity: Ask the patient to read small print (with glasses on, if they wear them) from a book or newspaper with one eye covered. Note any side to side differences. In a face-to-face consultation, the patient should read from a Snellen chart at 30–40 cm. In cases of severe visual impairment light perception should be tested using a small flashlight.

Field of vision: Ask the patient focus on a target in front of them, then hold out their arm to the side whilst wagging one finger. Ask them to move their arm toward the midline (whilst still wagging the finger), instruct them to tell you when they first see the movement of the finger. Repeat this on either side, at shoulder height, above the head and below the chin. Note any side to side differences or reduction in the visual field.

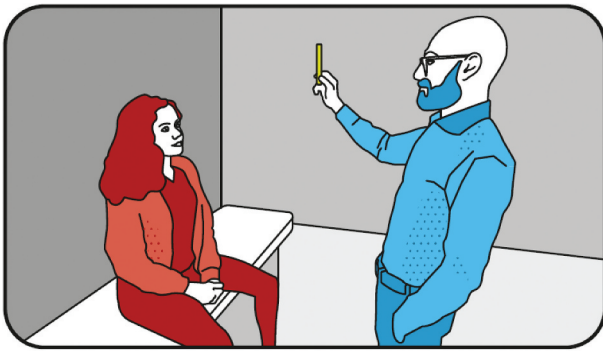


Figure 6. CN III, IV, VI test (H test for extra-ocular movement and control).

In a face-to-face consultation, the patient is instructed to look directly at the examiner's eye while the non-tested eye remains covered (see [Figure 4](#)). A red pen or the examiner's finger should be brought in from four directions diagonally toward the center of the visual field. The patient should state when the pen/finger becomes clearly detectable in order to detect any visual field deficits.

CN III – Oculomotor nerve

Ask the patient to outstretch their arm raise their finger, and then move it toward their nose, following with the eyes (accommodation or convergence), note symmetry of movement and side to side differences.

Pupillary reaction to light: In a dimly lit environment, ask the patient to shine a small flashlight one eye (close to the webcam). Note the reaction in the ipsilateral and contralateral eye. Both pupils should constrict in reaction to bright light. A direct response is the constriction that occurs when the pupil is exposed to light. The consensual or indirect response refers to the simultaneous constriction of the opposite pupil. Note any side to side differences. In a face-to-face consultation you can use a small flashlight to test the light reflex (see [Figure 5](#)).

CN's III, IV and VI, Oculomotor, Trochlear and Abducens nerves

Movement of the eyes (extra ocular movements): Ask the patient to draw a H shape 2–3 times with their finger and follow with their eyes.

In a face-to-face consultation, move a pen in an H pattern 30–40 cm in front of the patient. Patients should be asked to follow the target with their eyes without moving their head (see [Figure 6](#)). Observe the symmetry of movement in each eye, deviations, lag or nystagmus (an involuntary, rapid and repetitive movement of the eyes – either horizontal, vertical or rotary). Note any side to side differences and try to assess direction of deficit.

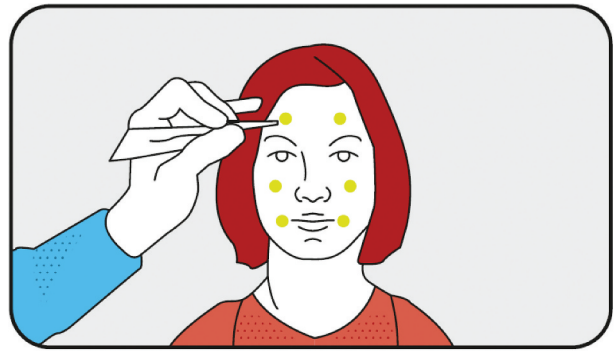


Figure 7. CN V test (sensory component).

Examination of the face, jaw, muscles of expression, throat and tongue – CN V, VII, IX, X, XII

Observation: Check for facial or neck/shoulder asymmetry. Note wasting, or any side to side differences.

CN V – Trigeminal nerve (sensory)

Check facial sensation – ask the patient to stroke their skin in the distribution of the ophthalmic (scalp, forehead, upper eyelid), maxillary (lower eyelid, cheek, upper lip/teeth) and mandibular divisions (chin jaw lower lip, mouth, lower teeth/gums).

In a face to face-to-face consultation, the sensory component is tested using a cotton wool ball and blunt tip needle sequentially, while comparing sides (see [Figure 7](#)). Note any side to side differences. The corneal reflex is tested with a wisp of cotton touching the cornea gently for triggering the blinking reflex of both eyes.

CN V – Trigeminal nerve (motor)

Test jaw strength – ask the patient to place a fist under their jaw and open their mouth against their own resistance. This is a very powerful movement in normal circumstances. Ask if it feels strong. Ask them to resist side to side movement of the jaw. Ask them to clench their teeth together. Observe and palpate (if face to face) the size and contraction of the masseter. Note any side to side differences.

In a face-to-face consultation, the motor component is assessed by examining the function of the temporalis, masseteric and pterygoid muscles.

CN VII – Facial nerve

Observation of a patient's face can yield the initial clues of asymmetrical expression. Ask patient to smile/frown, raise eyebrows, puff out cheeks. Check for symmetry. Ask about taste. In a face-to-face

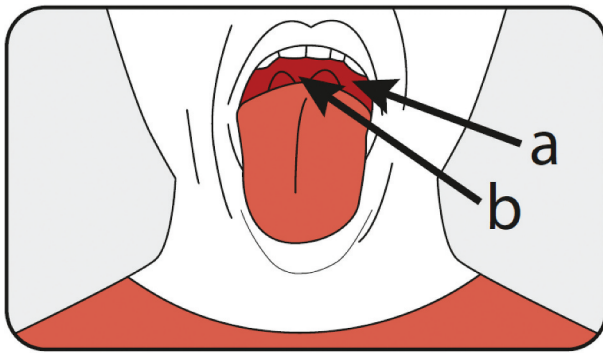


Figure 8. CN X test (observation of the soft palate [a] and uvula ([b])).

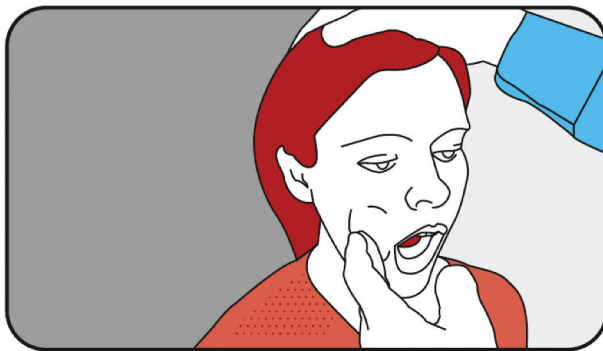


Figure 9. CN XII test (test tongue against resistance).

consultation, ask the patient to close both eyes tightly while you try to force open each eye to test strength. Then, assess the muscles of expression asking the patient to show the teeth, to 'puff out' the cheeks palpating them to determine any difference in tone.

CN IX – Glossopharyngeal nerve

Ask patient to swallow (a sip of water). Ask if swallowing feels normal or if they have noticed any difficulty with eating or drinking. In a face-to-face consultation, an

unilateral lesion in the glossopharyngeal nerve can manifest as loss of the ipsilateral gag reflex that is triggered with a tongue depressor touching gently the back of the throat on one side (tests CN IX and X together).

CN X – Vagus nerve

Ask patient to open mouth flatten tongue and say 'Aaaaaaaah', observe for symmetrical elevation of the soft palate and central ascent of the uvula. (see Figure 8).

CN IX and X

Ask the patient about their voice (hoarseness). Ask them if their cough sounds normal. In a face-to-face consultation, take note of the patient's voice during conversation for a hoarse voice or a 'bovine' sound (a non-explosive or hollow sound quality) during cough.

CN XII – Hypoglossal nerve

The tongue is carefully inspected for signs of atrophy, asymmetry or fasciculation. Patient protrudes tongue, check for deviation. Look for fasciculations (visible spontaneous and intermittent muscle contractions). Ask the patient to push out their cheek with their tongue, and check power by pushing against outside of cheek. Note any side to side differences.

In a face-to-face consult, power is examined by having the patient press the tip of the tongue against each cheek while the examiner tries to dislodge it (Figure 9).

Head neck and shoulder motor function



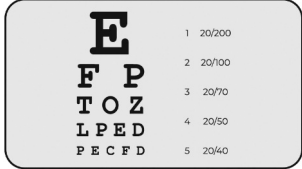



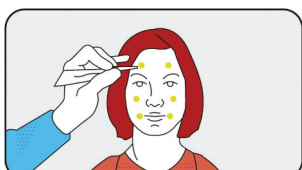

CN XI – Accessory nerve

Ask the patient to resist head rotation (sternocleidomastoid) and shrugging of shoulders (upper fibers trapezius).

Table 3. CN subjective questions (Diagonal lines – Sensory function – Smell/Hearing; Horizontal lines – Motor and sensory function of the eyes; Vertical lines – Motor and sensory function of the face/jaw/throat/tongue; Crossed lines – Motor function of the head/neck/shoulders).


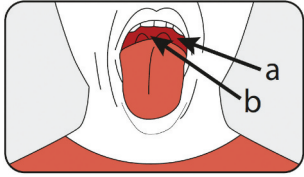
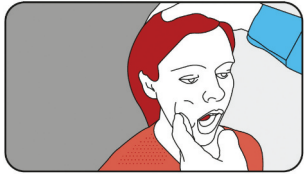

Number	Name	Subjective Examination Questions
I	Olfactory	Have you noticed any recent changes of the ability to smell?
VIII	Vestibulocochlear	Have you noticed any recent alteration to your hearing? Any balance issues, motion sickness or tinnitus linked to eye movements?
II	Optic	Have you noticed any recent difficult reading or alteration to your vision? Have your extremes/fields of vision altered?
III & VI	Oculomotor & Abducens	Have you noticed any recent alteration to your vision?
IV	Trochlear	Have you noticed any recent alteration to your vision or unsteadiness?
V	Trigeminal	Have you noticed any recent alteration to your ability to eat or chew? Have you noticed any recent alteration to your facial sensation?
VII	Facial	Have you noticed any recent alteration to your facial features e.g. smile? Any recent alteration to taste?
IX & X	Glossopharyngeal & Vagus	Have you noticed any recent alteration to eating, taste or ability to swallow. Does your cough sound the same as usual? Any change in the sound of your voice or hoarseness?
XII	Hypoglossal	Have you noticed any recent alteration to eating, swallowing, speech (articulation) or tongue function?
XI	Accessory	Have you noticed any recent alteration to your head neck or shoulder function?

Table 4. Cranial nerves, their functions and examination (Diagonal lines – Sensory function – Smell/Hearing; Horizontal lines – Motor and sensory function of the eyes; Vertical lines – Motor and sensory function of the face/jaw/throat/tongue; Crossed lines – Motor function of the head/neck/shoulders).

Number/Name	Function	Examination	
I Olfactory	Smell (olfaction)	Identify a familiar smell (soap/perfume)	
VIII Vestibulocochlear (auditory)	Hearing, balance/equilibrium	Ask patient if they can hear fingers rubbing (close to ear) or whispered number sequence	
II Optic	Vision (acuity and field)	Test each eye with Snellen chart or newspaper. Test visual fields in 4 quadrants	
III Oculomotor	Eye movements, elevation of eyelid. Pupil size and reactivity to light	Check pupil reaction to light (both should constrict). Check all EOMS (H-test). Check accommodation (finger to nose)	
IV Trochlear	Eye movement (vertical and adduction)	H-Test – observe down and in	
VI Abducens	Eye movements – abduction	H-Test – observe side to side	
V Trigeminal	Chewing, face/mouth sensation. Corneal reflex (sensory)	Test jaw strength (open mouth) – try to close/move laterally Check facial sensation – sharp/blunt	
VII Facial	Facial expression, eyelid and lip closure, taste. Corneal reflex (motor)	Ask patient to smile, raise eyebrows, puff out cheeks. Check for symmetry. Ask about taste.	

(Continued)

Table 4. (Continued).

Number/Name	Function	Examination	
IX Glossopharyngeal	Gagging, swallowing (sensory), taste	Assess gag reflex with tongue depressor. Ask patient to swallow.	
X Vagus	Gagging, swallowing (motor), speech (sound)	Ask patient to say 'Aaaaaaaah', observe for symmetrical elevation of palate [a] and uvula [b].	
XII Hypoglossal	Tongue movement, speech (articulation)	Patient protrudes tongue, check for deviation, look for fasciculations. Patient pushes out cheek with tongue, check power by pushing cheek.	
XI Accessory	Head/neck/shoulder movement	Check resisted head rotation (sternocleidomastoid) and shoulder elevation (trapezius – upper fibers).	

In a face-to-face consultation, test power manually. Note any side to side differences.

Interpretation of findings

Clinicians should recognize the need to test CN function when patients present with complex or confusing presentations which may be worsening or unresponsive to management. If the subjective examination (see Table 3.) raises the index of suspicion and the CN examination reveals side to side differences or abnormal responses, which may or may not fit with the overall clinical picture, then this would be an indication to refer on for further examination or appropriate imaging/testing.

The urgency of referral will always very much depend on the full clinical picture and the status of the patient's symptoms (e.g. static or worsening). Ideally, urgent referral should be made using the situation, background, assessment, recommendation (SBAR) method [15] or appropriate variant which is a communication technique that increases patient safety and is current 'best practice' to deliver information in critical situations [16].

It is however, important that clinicians are aware that there is little or no specific data available to support the diagnostic accuracy (i.e. validity and reliability) of a complete CN examination [2]. However, psychometrics from elements of cranial nerve examination support at least moderate reliability and validity of cranial nerve examination [17]. This may link to the wide variety and combinations of potential pathologies that may lead ultimately to neural impairments. These impairments may manifest in a variety of presentations encountered by musculoskeletal clinicians and may be detected via appropriate examination (as an example a case study is presented in Appendix 3). An comprehensive overview of the CN, their functions and examination is presented in Table 4.

Conclusion

CN examination is an important component of the skill set of physiotherapists working in first contact settings where they may encounter patients with neck pain, orofacial pain, headache and associated symptoms. Gaining

CRANIAL NERVE EXAMINATION

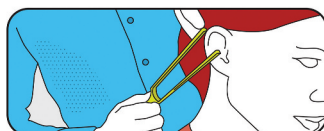
Ask the patient to close both eyes tightly while the examiner attempts to force open each eye to test strength. Then, assess the muscles of expression asking the patient to show the teeth, to “puff out” the cheeks palpating them to determine any difference in tone.



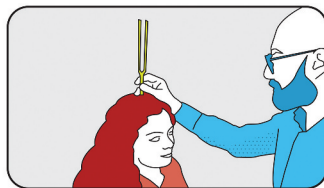
Vestibulocochlear Nerve (CN VIII)



Auditory – Hearing: the examiner vigorously rubs the fingers together near to each ear having the patient closing the eyes. If there is lateralization or hearing abnormalities perform the Rinne and Weber tests using the 256-Hz tuning fork;



Rinne's test: The tuning fork is struck and placed on the mastoid process. The patient is requested to indicate when the sound is no longer audible. As soon as the sound is extinguished, the tuning fork is placed next to the external auditory meatus to assess whether it can be heard. In a patient with normal hearing, air conduction should be greater than bone conduction, so the patient should be able to hear the tuning fork;



Weber's test: place the tuning fork in the middle of the forehead and the sound is heard from there. The sound should be heard equally on both ears;

Vestibular system: perform specialized tests such as Hallpike's maneuver or assessing the patient's gait and looking for nystagmus

Glossopharyngeal Nerve (CN IX)

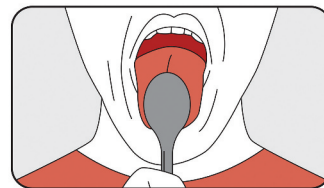


Clinical examination of the glossopharyngeal nerve is typically performed in conjunction with the vagus nerve, as separate testing is challenging. A unilateral lesion in the glossopharyngeal nerve can manifest as loss of the ipsilateral gag reflex that is triggered with a tongue depressor touching gently the back of the throat on one side.

Vagus Nerve (CN X)



The vagus nerve is typically evaluated in conjunction with the glossopharyngeal nerve. The examiner should take note of the patient's voice during conversation for a hoarse voice or a hollow “bovine” sound during cough;



Examine the position of the uvula and look for symmetrical elevation of the soft palate having the patient saying “ahhh” while depressing the tongue;

Assess bedside swallowing by asking the patient to drink small sips of water.

Spinal Accessory Nerve (CN XI)



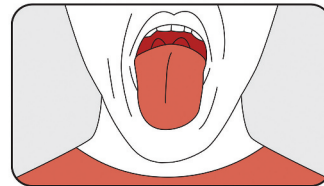
Test the trapezius muscle by asking the patient to shrug their shoulders while applying resistance;



Test the sternocleidomastoid muscle by asking the patient to turn the head against resistance.

Hypoglossal Nerve (CN XII)

The tongue is carefully inspected for signs of atrophy, asymmetry or fasciculation;



Power is examined by having the patient press the tip of the tongue against each cheek while the examiner tries to dislodge it.

CRANIAL NERVE EXAMINATION

how to read
the tests

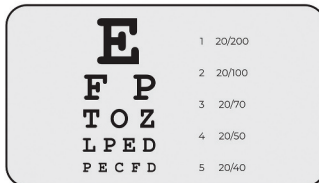


CN I - Olfactory nerve



Screen for Anosmia: note that anosmic patients do not always complain about loss of smell, rather than altered taste. One nostril should be occluded while the examiner asks the patient to smell commonly items such as coffee and soap with each nostril.

CN II - Optic nerve



Visual acuity: the patient should read through a Snellen chart at 30-40 cm.

During the examination, one eye should be completely covered. In cases of severe visual impairment light perception should be tested using a pen torch;



Visual fields: The patient is instructed to look directly at the examiner's eye while the non-tested eye remains covered.

A red pen or the examiner's finger should be brought in from four directions diagonally towards the center of the visual field.

The patient should state when the pen / finger becomes clearly detectable in order to detect any visual field deficits;

Fundoscopy: use the ophthalmoscope to assess the optic disc and retina while the patient is looking into the distance.

Oculomotor, Trochlear, and Abducens Nerves (CN III, IV, and VI)

Pupils: Inspect the size, shape, and symmetry of pupils;



Test the light reflex: a direct response is the constriction that occurs when the pupil is exposed to light.

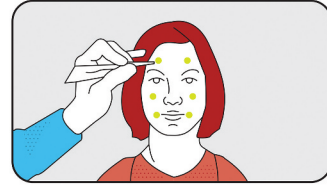
The consensual or indirect response refers to the simultaneous constriction of the opposite pupil;

Test the accommodation asking the patient to focus on the nose;



Eye Movements: Assesses failure of movement, diplopia and nystagmus. The examiner should be 30-40 cm in front of the patient moving a hat pin in an "H" pattern. Patients should be asked to follow the target with their eyes without moving their heads.

Trigeminal Nerve (CN V)



The sensory component is tested using a cotton wool ball and blunt tip needle sequentially on the forehead, malar eminence and lower face over the mandible, while comparing sides;



Corneal reflex is tested with a wisp of cotton touching the cornea gently for triggering the blinking reflex of both eyes;



The motor component is assessed by examining the function of the temporalis, masseteric and pterygoid muscles.

Facial Nerve (CN VII)

Observation of a patient's face can yield the initial clues of asymmetrical expression;



knowledge and understanding of the CN's function and potential reasons for impairment is likely to increase the performance of testing. This will allow clinicians to pick up on subtle clues that may be offered by patients during the subjective examination (e.g. difficulty swallowing, loss of taste or smell, etc.) and lead them to targeted physical examination and appropriate triage.

This article illustrates that CN testing can be performed logically, efficiently and quickly. Clinicians who treat patients who present with neck/head/orofacial pain and associated symptoms should develop the ability to perform and interpret CN examination and know when to refer to medical colleagues as indicated by the results of the examination.

Conflicts of interest

No potential conflict of interest was reported by the author(s).

Notes on contributor

Alan Taylor is a physiotherapist, educator and writer. He is the author of over 30 peer-reviewed articles (primarily cervical arterial dysfunction (CAD)/haemodynamics/Sports injury), and 5 book chapters (Haemodynamics/CAD/Clinical Reasoning/Manual Therapy). He has devised and taught over 100 one-day and two-day courses in these areas. He has been a regular key note speaker at numerous courses and conferences in the UK and Worldwide since 2002. He also works as an 'expert witness' in the field of medical negligence cases, related to altered haemodynamics and arterial dysfunctions.

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Appendix 1: Cranial nerve functions and dysfunctions

Cranial nerve I – Olfactory nerve

Type: Sensory

Function: Smell

The afferent nerve fibers of the olfactory receptors transmit nerve impulses about odors to the CNS (olfaction).

Mechanisms: Loss of smell is known as anosmia.

Anosmia is well-established sequela of head injury – with or without skull fracture.

May follow blunt trauma such as a road traffic collision or a head injury in sport. Professional boxers or fighters may suffer anosmia as a result repeated trauma to the head [18]. Temporary anosmia may result from inflammatory responses in relation to infection.

Note: Covid19 is a common feature of temporary anosmia.

Cranial nerve II – Optic nerve

Type: Sensory

Function: Vision (acuity and field)

Paired nerves that transmit visual information from the retina (back of the eye) to the brain.

Mechanisms: Optic neuritis or inflammation can cause damage to the protective sheath (myelin) surrounding this nerve and the nerve itself. May affect one OR both eyes – common in people who have multiple sclerosis (MS).

Glaucoma may affect the optic nerve due to high intra-ocular pressure, which compresses the optic nerve and causes cells to die. It is referred to as atrophy of the optic nerve.

Nutritional optic neuropathy is a cause of bilateral, symmetrical, and progressive visual impairment with loss of central visual acuity. This mechanism is considered increasingly common due to the widespread use of bariatric surgery and strict vegetarian or vegan diets.

Ischemic optic neuropathy due to reduced blood flow to the optic nerve, may be linked to atherosclerosis, or arteritis e.g. Giant cell arteritis (GCA) a condition commonly presenting as headache.

Cranial nerve III – Oculomotor nerve

Type: Motor

Function: Eye movement: Innervates extrinsic eye muscles that enable most movements of the eye and that raise the eyelid.

Mechanisms: Disorders of the oculomotor nerves, can impair ocular motility, pupillary function, or both. Symptoms and signs include diplopia and ptosis (drooping of the upper eyelid). The pupil may be dilated, and light reflexes impaired. The affected eye may deviate slightly out and down in straight-ahead gaze; adduction is slow and may not proceed past midline. There may also be paresis of eye adduction and of upward and downward gaze. When downward gaze is attempted, the superior oblique muscle may cause the eye to adduct slightly and rotate.

Neuropathy may be linked to trauma, small vessel disease (older adults) or space occupying lesions e.g. aneurysm or neoplasm.

Cranial nerve IV – Trochlear nerve

Type: Motor

Function: Eye movement

Mechanisms: May affect one or both eyes.

Nerve palsy impairs the superior oblique muscle, causing paresis of vertical gaze, mainly in adduction. Patients report seeing double images, one above and slightly to the side of the other, and the eyes do not adduct normally. This may affect every day function e.g. walking in crowds or going downstairs.

Possible causes are closed head injury (common), which may cause unilateral or bilateral palsies, infarction due to small vessel disease (e.g. in diabetes). Rarely, this palsy may result from aneurysms, tumors, or MS (Kung and Van Stavern, 2015).

Cranial Nerve V – Trigeminal nerve

Type: Motor and sensory

Function: Somatosensory information (touch, pain) from the face and head;

Motor function – chewing. Reflex: sensory element of corneal reflex (rarely used)

Mechanisms: Commonly manifests as severe facial pain and allodynia.

Possible causes may be compression of the trigeminal nerve at its root by an aberrant loop of an intracranial artery (e.g. anterior inferior cerebellar artery or dilated basilar artery). Rarely, a venous loop may compress the nerve at its root entry zone into the brain stem. Less common causes include compression by tumors, arteriovenous malformation, aneurysm, and MS (Khan et al., 2017).

Cranial nerve VI – Abducens nerve

Type: Motor

Function: Eye movements (abduction). Palsy causes impaired abduction and horizontal diplopia

Mechanisms: Palsy affects the lateral rectus muscle, impairing eye abduction and may cause severe head pain. The eye may be slightly adducted when the patient looks straight ahead. May be secondary to nerve infarction, encephalopathy, trauma, infection, increased intracranial pressure, vasculitis, or may be idiopathic (Kung and Van Stavern, 2015).

Cranial nerve VII – Facial nerve

Type: Motor and sensory

Function: Taste (anterior 2/3 of tongue); somatosensory information from ear; controls muscles of facial expression. Motor element of corneal reflex.

Mechanisms: Sudden onset, unilateral peripheral facial nerve palsy (Bell's Palsy).

Symptoms are hemi-facial paresis of the upper and lower face. The mechanism is thought to be swelling of the facial nerve due to an immune or viral disorder.

The swollen nerve is maximally compressed as it passes through the labyrinthine portion of the facial canal, resulting in ischemia and paresis. Current evidence suggests that common viral causes are: Herpes simplex virus infection (most common), Herpes zoster. Other viral causes may include coxsackievirus, cytomegalovirus, adenovirus, Epstein-Barr, mumps, rubella, and influenza B viruses.

Cranial nerve VIII – Vestibulocochlear nerve

Type: Sensory

Function: Hearing and balance

Mechanisms: This is the nerve along which the sensory cells (the hair cells) of the inner ear transmit information to the brain. This facilitates hearing and equilibrium.

Dysfunction of the nerve is thought to be linked to viral infections and may cause a range of symptoms including: hearing loss, vertigo, false sense of motion, loss of equilibrium (in dark places), nystagmus, motion sickness or gaze-evoked tinnitus.

Cranial nerve IX – Glossopharyngeal nerve

Type: Motor and sensory

Function: Taste (posterior 1/3 of tongue); Somatosensory information from tongue, tonsil, pharynx; control of some of the muscles used in swallowing.

Mechanisms: Characterized by episodes of unilateral brief, excruciating pain which occur spontaneously or are precipitated by stimulation of the nerve by chewing, swallowing, coughing, talking, yawning, or sneezing. The pain usually begins in the tonsillar region or at the base of the tongue and may radiate to the ipsilateral ear. There may be difficulty swallowing; impairment of taste over the posterior 1/3 of the tongue and palate; impaired sensation over the posterior 1/3 of the tongue, palate, and pharynx; absent gag reflex.

Dysfunction may result from nerve compression by an aberrant, pulsating artery. Rarely, the cause is a tumor in the neck, peritonsillar abscess, carotid aneurysm/dissection, or a demyelinating disorder.

Cranial nerve X – Vagus nerve

Type: Motor and sensory

Function: Sensory, motor and autonomic function of viscera (glands, digestion, heart rate)

Mechanisms: Vagus nerve lesions produce palatal and pharyngeal paralysis/laryngeal paralysis. This is characterized by a loss of reflex contraction of the palate or altered gag reflex, hoarseness of the voice, which is sometimes associated with an abnormal cough. There may be abnormalities of esophageal motility, gastric acid secretion, gallbladder emptying, altered heart rate; and other autonomic dysfunctions. This nerve runs part of its course within the carotid

sheath and may be implicated in aneurysm or dissection (Gutierrez et al., 2020).

Cranial nerve XI – Accessory nerve

Type: Motor

Function: Motor function – head/neck/shoulder

Mechanisms: Dysfunction results in weakness of the sternocleidomastoid muscle and upper portion of the trapezius muscle. Sufferers may exhibit signs of diminished muscle mass, fasciculations, and partial paralysis of the SCM and trapezius muscles, resulting in an asymmetric neckline. Weakness and atrophy of the upper portion of the trapezius muscle can produce a drooping shoulder, winged scapula, and a weakness of forward elevation of the shoulder. This may be secondary to neurogenic aetiology, as the nerve is vulnerable along its superficial course in the posterior triangle of the neck. Injury can occur secondary to blunt or direct trauma during sports injuries (Bordoni et al., 2020). Aneurysms of the internal carotid artery or a fracture to the atlas bone may compress the nerve leading to ischaemic damage. Additionally, Eagle-syndrome has been reported to negatively affect nerve function (Bordoni et al., 2020). Surgical procedures are also reported as a cause of injury to the spinal accessory nerve (Gutierrez et al., 2020).

Cranial nerve XII – Hypoglossal nerve

Type: Motor

Function: Movement and function of the tongue

Mechanisms: A clinical feature may be muscle atrophy and tongue weakness. Dysfunction is characterised by flaccid paralysis/weakness of the ipsilateral tongue musculature. An attempt to protrude the tongue results in deviation of the tongue toward the weak side because of the unopposed actions of the intact genioglossus muscle. As impairment of CN XII progresses, fasciculations (involuntary muscle contraction) can be seen on the ipsilateral tongue (Gutierrez et al., 2020).

Appendix 3. Case study.

Patient information

A 44 year old male hobby cyclist (former professional) attended his regular physiotherapy clinic, 24 hours after sustaining an acute onset, of right sided ear pain (described like 'someone hammering a nail into his ear'), fronto-lateral headache, momentary double vision and mild neck pain. He reported that the onset was whilst out cycling (during an intense hill sprint). He was fit and healthy, a regular exerciser and was on no medications. He had suffered a previous skull fracture sustained during competition 15 years previously, but had made a full recovery. He denied suffering headaches and reported that he had never suffered this type of pain before. His main ongoing symptom was an unfamiliar fronto-lateral headache which was resistant to self-medication (paracetamol and Ibuprofen).

He lived with his partner and was the proprietor of a cycle shop. He had attended for assessment because he was hoping to ride a 100 mile charity cycle ride in one weeks' time. On further specific questioning (see [Table 3](#)) he said that his episode of dizziness lasted "for a matter of seconds only". He did however, report some minor difficulties with swallowing, and a 'sore throat' that was affecting his voice which he did not associate with his presentation.

Physical examination

Objectively, there was observable muscle spasm on the right side of the neck.

Based on the unusual presentation (i.e. exercise induced, acute unfamiliar headache, medication resistance, episode of visual disturbance, subjective swallowing difficulties), and in line with the IFOMPT framework (IFOMPT, 2020), the order of the examination was modified to commence with vital signs, blood pressure and cranial nerve testing.

The findings were as follows:

Vital signs:

Pulse was 55 bpm and regular.

Temperature was normal.

Blood pressure (BP) taken on the left was 210/105 mm hg (1), 205/100 mm hg (2) and on the right 200/105 mm hg.

A full CN examination was unremarkable, until the lower nerves revealed asymmetrical elevation of the soft palate with deviation of the uvula toward the left. There was observable difficulty with swallowing a small amount of water.

No further examination was performed.

Analysis

The elevated blood pressure and cranial nerve findings were suggestive of a potentially serious pathology. The patient was triaged immediately from the clinic to accident and emergency (A&E) via the SBAR method detailing the objective findings and making a recommendation for further examination/ tests and a reported suspicion of impending stroke.

Follow-up

The patient attended A&E and, an hour later and was admitted to hospital, where MRA and Duplex US revealed the presence of a right sided carotid artery dissection. The vascular team were of the opinion that this was an exercise-induced dissection and may have been linked to his (previously unknown) elevated blood pressure. The vagus nerve (X) and the glossopharyngeal nerve (IX) both pierce the superior part of the carotid sheath (Garner and Baker, 2019) and may have been affected by local dilation of the vessel in response to the vascular insult. The patient was medicated and made a recovery. He has returned to leisure cycling but on the advice of the vascular team, limits his extreme efforts with the use of a pulse monitor alarm.

Discussion

The described case illustrates a practical application of CN testing. It is arguable that the BP findings alone were enough to triage the patient. However, the patient's report of difficulty swallowing (see [Table 3](#)) was a cue to consider testing the CN's, and allowed the referrer to offer a much clearer referral, which both offered anatomical clues to the potential pathology and was suggestive of urgency. Notably, there are no standard rules or interview items able to indicate when to perform CN examination and their testing rely on the practitioners clinical reasoning skills, knowledge of risk factor/red flags, and pathophysiology knowledge (IFOMPT, 2020).